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Miran Jang
Purdue University

Yuan Zhang
Purdue University

Juan Bai
Purdue University

An Jun-Bae
Seowon University

Gun-Hee Kim
Duksung Women's University

See next page for additional authors

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Piceatannol, a Natural Stilbene, Extends the Lifespan of Caenorhabditis elegans Under Fasting Through Inhibition of Lipolysis

Miran Jang[1], Yuan Zhang[1], Juan Bai[1], An Jun-Bae[2], Yeonhwa Park[3], Gun-Hee Kim[4], and Kee-Hong Kim[1]

[1]Purdue University; [2]Seowon University; [3]University of Massachusetts Amherst; and [4]Duksung Women's University

Objectives: Lipolysis is the catabolic process that hydrolyzes triglyceride (TG) to free fatty acids (FFAs) and glycerol under negative energy balance such as fasting. In adipocytes, adipose TG lipase (ATGL), hormone-sensitive lipase (HSL), and monoglyceride lipase play key roles in a series of TG hydrolysis reactions in mammals. However, overly activated adipose lipolysis is believed to contribute to link between obesity and systemic inflammation and oxidative stress. We previously demonstrated that piceatannol (PIC), a natural resveratrol analogue, inhibits adipogenesis in cultured adipocytes and lipogenesis in *Caenorhabditis elegans*. Furthermore, we showed that PIC extends the lifespan of *C. elegans* via the insulin/IGF-1 signaling. However, the effects of PIC on lipid metabolism during fasting state is unknown.

Methods: We conducted Oil-Red-O assay, Enzyme assay (TG and Free glycerol contents), PCR analysis and lifespan assay.

Results: In this study, we demonstrated that PIC-treated *C. elegans* exhibited suppressed lipolysis under fasting as judged by increased lipid accumulation and TG levels with decreased free glycerol level. Consistent with these findings, PIC treatment resulted in decreased mRNA levels of genes involved lipolysis such as *atgl-1, hosl-1* and *aak-2* in fasted *C. elegans*. Also, PIC treatment augmented fasting-induced lifespan of *C. elegans* by an increased *daf-16* gene expression. However, such effect was abolished when *atgl-1*, *aak-2*, and *daf-16* mutants were treated with PIC. In addition, we also found that autophagy is required for PIC-induced lifespan in *C. elegans* during fasting since autophagy inhibitor treatments and autophagy gene deficient worms resulted in blunting the lifespan extension effect of PIC.

Conclusions: Collectively, our results indicate that PIC contributes to lifespan extension in *C. elegans* during fasting possibly through regulating lipolysis- and/or autophagy-dependent lipid metabolism.

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